Claims

What is claimed is:

1. A method for enhancing recovery by epithelial cells from ischemia by targeting distinct lesions, comprising:

inhibiting internalization of intercellular junctions, E-cadherin, occludin or other membrane proteins;

promoting reuse of preexisting components by targeting for activation specific signaling events during short-term ischemia;

inhibiting degradation of E-cadherin or other key proteins necessary for the maintenance of the polarized epithelial cell phenotype; and

enhancing the protein folding and assembly capacity in the ER and/or cytosol with agents which upregulate cytoprotective chaperones,

wherein the enhancing helps to reconstruct degraded adherens and tight junctions by *de novo* synthesis and movement of membrane proteins, and alleviation of cellular stress by raising levels of molecular chaperones.

2. The method according to claim 1, wherein the inhibiting of the internalization requires early intervention with drugs or growth factors that specifically modulate signaling through IP_3-sensitive calcium stores, G-proteins, protein kinase C, and other kinases all of which are implicated in the reassembly response during the calcium switch.

3. The method according to claim 1, wherein the promoting refers to facilitating the resorting of growth factor receptors to the cell surface through modulation of signaling pathways to enhance the effectiveness of endogenous and/or exogenous growth factors administered after ischemic insult.

4. The method according to claim 1, wherein the inhibiting degradation refers to prevention of proteolytic clipping of key proteins.

5. The method according to claim 1, wherein the agents which upregulate cytoprotective chaperones comprise inhibitors of proteasome.

6. The method according to claim 1, wherein the agents which upregulate cytoprotective chaperones comprises pretreatment with tunicamycin.